Paradoxically, however, it appears to have a hypercoagulable effect. There are at least two explanations for the latter effect. The lupus anticoagulant reacts with platelet wall phospholipids (platelet factor 3) (Thiagarajan et al. 1980), damaging them and increasing their adhesiveness and thereby initiating thrombosis. This may also explain the accompanying thrombocytopenia. Carreras et al. (1981a) found that the lupus anticoagulant inhibited the production of prostacyclin in the pregnant uterus, possibly by interfering with the release of arachidonic acid. This leads to placental thrombosis and hence intrauterine death.

We conclude that a search for anticardiolipin antibodies and for lupus anticoagulant is indicated in patients with venous or arterial thrombosis, pulmonary hypertension (Asherson et al. 1983), thrombocytopenia or repeated intrauterine fetal death even in the absence of SLE or 'lupus-like' disorders (Lancet 1984).

## Acknowledgment:

We are grateful to Dr G R V Hughes and his colleagues at the Hammersmith Hospital, London, for measuring the anticardiolipin antibodies in this patient.

#### References

(1983) British Medical Journal 287, 1024-1025. Boey M L, Colaco C B, Gharavi A E, Elkon K B, Loizou S & Hughes G R V (1983) British Medical Journal 287, 1021-1023 Bowie E J W, Thompson J H, Pascuzzi C A & Owen C A (1963) Journal of Clinical Investigation 62, 416-430 Carreras L O, Machin S J, Deman R et al. (1981a) Lancet i, 224-246 Carreras L O, Vermylen J, Spitz B & Van Assche A (1981b) British Journal of Obstetrics and Gynaecology 88, Exner T, Rickard K A & Kronenberg H (1978) British Journal of Haematology 40, 143-151 Firkin B G, Howard M A & Radford N (1980) Lancet ii. 336 Green D, Hougie C, Kazmier F J et al. (1983) Thrombosis and Haemostasis 49, 144-146 Harris E N, Gharavi A E, Boey M L et al. (1983) Lancet ii, 1211-1214 Lancet (1984) i, 1157-1158 Landi G, Calloni M V, Sabbadini M G, Mannucci P M & Candelisle L (1983) Stroke 14, 377-379 Lubbe W F, Butler W S, Palmer S J & Liggins G C (1983) Lancet i, 1361-1363 Much J R. Herbst K D & Rapaport S I (1980) Annals of Internal Medicine 92, 156-159 Nilsson I M, Astedt B, Hedner U & Berezin D (1975) Acta Medica Scandinavia 197, 153-159 Solier R P & Boffa M C (1980) Nouvelle Presse Medicale (Paris) 9, 244-246 Tan E M, Cohen A S, Fries J F et al. (1982) Arthritis and Rheumatism 25, 1271-1277 Thiagarajan P, Shapiro S S & De Marco I (1980) Journal of Clinical Investigation 66, 397-405

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# Rhinophyma<sup>1</sup>

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Rhinophyma is a relatively common acquired deformity of the nose, the treatment of which has caused much dispute. A case is reported of a huge rhinophyma which was treated successfully by surgical shaving and spontaneous re-epithelialization.

# Case report

A 73-year-old man presented with a huge rhinophyma (Figure 1A) which had started as a gradually increasing thickening of the nasal skin two years previously. An enormous increase in size over the six months prior to presentation had

<sup>1</sup>Case presented to Sections of Dermatology and Plastic Surgery, 16 February 1984. Accepted 21 March 1985 caused such cosmetic embarrassment that the patient was living in total isolation. In addition the heavy, pendulous growth acted as a valve, obstructing the nares and causing breathing difficulty (Figure 1B). Under general anaesthetic the nose was injected with local anaesthetic containing adrenaline to reduce bleeding. The growth was then shaved off using a scalpel. At the end of the procedure haemostatic foam was applied to encourage haemostasis and provide a dressing. This separated spontaneously at six days leaving a clean, dry wound (Figure 1c) which epithelialized completely within two weeks. The final result was both functionally and cosmetically acceptable to the patient (Figure 1D).

### Discussion

Rhinophyma is a hypertrophy of the skin of the lower half of the nose which develops over a period of years. The resulting lesion varies in size from mild localized or generalized overgrowth to a large, bulbous and discoloured mass.

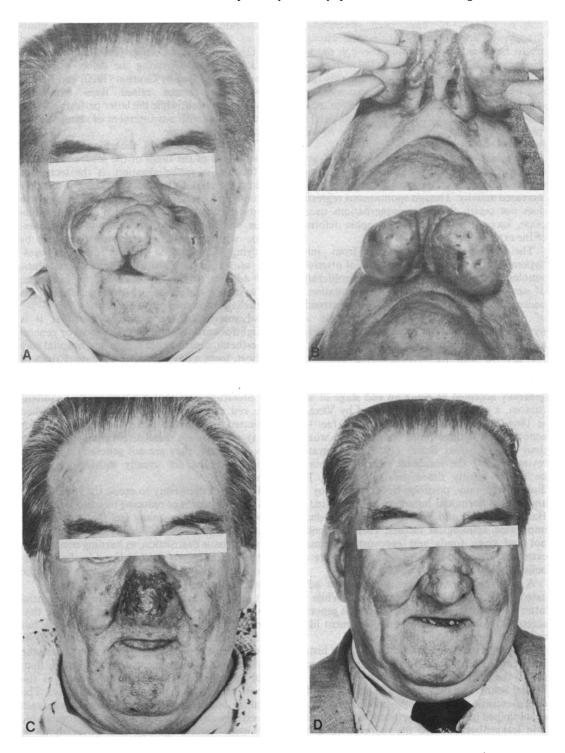


Figure 1. A and B: preoperative appearance of huge rhinophyma, showing valvular obstruction of the nares and the sulcus dividing the rhinophymatous from the normal tissue. C: at six days, the clean, dry wound after spontaneous separation of the dressing, showing epithelialization to be well advanced. D: satisfactory cosmetic result two months postoperatively.

It is reputed to have been described by Hippocrates and the early Arabs, and examples are to be found in portraiture through the ages (Hollaender 1903, 1905). Hebra (1845) first designated it rhinophyma from the Greek rhis (nose) and phyma (growth). There are a vast number of synonyms for the condition, ranging from 'potato nose' and 'rum blossom' to 'pachydermatosis' and 'pseudoelephantiasis of the nose', over 30 being listed by Odou & Odou (1961).

The disease progresses by a series of acute exacerbations and remissions but with persistence of the cutaneous overgrowth between bursts of increased activity. Though spontaneous regression does not occur, the acute exacerbations usually cease, leaving the typical hypertrophic deformity of the external nose.

The microscopic progress is from initial hyperaemia to permanent dilatation of arterioles, venules and capillaries, along with proliferation of new blood vessels. There is transudation of oedema fluid resulting in fibrosis and obstruction of the sebaceous glands. These dilate and hypertrophy, producing sebum-filled cysts which give the characteristic nodular appearance to the growth and its accompanying unpleasant odour.

The aetiology of the disease is unknown but is generally accepted as being an end stage of acne rosacea, a connection first suggested by Virchow in 1864. Against this hypothesis is the fact that acne rosacea is three times commoner in women than men, whereas Smith (1958), by literature review, found the incidence of rhinophyma to be nearly twenty times greater in men. This discrepancy may possibly be explained by an androgenic hormonal influence, as postulated by Hamilton (1941) and Wolfe (1943). Traditionally implicated aetiological factors, such as alcohol, hot and spicy foods and exposure to climatic extremes, are not consistent findings and are not believed to be involved.

A wide range of treatments have been advocated, both surgical and non-surgical. While the latter may have a place in early cases, it is generally accepted that surgery is the only treatment likely to benefit the established rhinophyma.

The surgery of this condition has a long history and remains controversial. According to Joseph (1931) the first operation for rhinophyma was by Daniel Sennert in 1629. In 1845 Dieffenbach described excision of the rhinophymatous tissue via a cross-shaped incision with primary wound closure. Von Langenbeck excised the tissue down to the cartilaginous base, leaving the wound to heal by granulation; and in 1864 Stromeyer advocated shaving off the rhinophyma with preservation of the fundi of the sebaceous glands as foci for re-epithelialization, a technique later termed de-

cortication (Matton et al. 1962). Split-skin grafting of the defect was recommended by Wood (1912), whereas MacComber (1946) favoured full-thickness grafts. Covering the defect with local flaps was described by Gratton (1920) and Berson (1948); the former raised flaps from the rhinophyma itself, while the latter performed complete excision with advancement of skin from the upper part of the nose.

Currently, shaving off the rhinophyma, with perservation of the fundi of the sebaceous glands as foci for re-epithelialization, is the technique most often used. Care must be taken not to shave beyond the depth of the sebaceous glands nor to expose cartilage. Bleeding, which can be trouble-some, can be reduced by injection of local anaesthetic with adrenaline before starting, and by applying a haemostatic gauze or foam as a dressing at the end. This encourages haemostasis and adheres to the raw area by the formation of a crusty coagulum which separates off as epithelialization progresses beneath.

Advantages of the technique are that it is straightforward to perform under local or general anaesthesia, dressings are simple and hospital stay is short, from a day case to a few days. Critics of the technique claim that there is a risk of recurrent disease, though there are but few reported cases in the literature, and that a cosmetically unacceptable, red, shiny scar, which may be unstable when subjected to climatic extremes, can persist for many years. While these disadvantages undoubtedly can occur, they are not generally a problem and the results are usually satisfactory to all concerned.

The use of diathermy to excise the rhinophyma has the sole benefit of reducing bleeding during the procedure. However, many feel that this is more than outweighed by the difficulty in controlling the depth of tissue destruction, as burning occurs deep to the resection level, with delayed healing, cartilage destruction and nasal distortion being the possible sequelae (Maliniac 1931).

It was in attempts to overcome the potential problems of the re-epithelialization technique that skin grafting and local flaps were advocated; but both of these have drawbacks. Skin grafting is more difficult to perform, the graft may fail to take or its colour and texture match may be less than ideal and there is the additional wound of the donor area with its possible morbidity. The majority of authors feel that these disadvantages exceed the claimed advantages of a more stable and more natural looking skin cover, both of which have been frequently disputed. The use of local flaps is almost universally criticized as it must inevitably employ diseased skin, except in the most localized of cases. The technique is now effectively obsolete.

#### References

Berson M I

(1948) Journal of Plastic and Reconstructive Surgery 3, 740-744

(1845) Operative Chirurgie. F A Brockhaus, Leipzig

(1920) Journal of the American Medical Association 74, 1450

Hamilton J R

(1941) Journal of Clinical Endocrinology 1, 570-592

Hebra J F

(1845) Zietschrift der k k Gesellschaft der Aertze zu Wein Zweiter Jahrg 1, 148, 211

Hollaender E

(1903) Die Medizin in der Klassischen Malerei. Ferdinand Enke Verlag, Stuttgart

(1905) Die Karikatur und Satire in der Medizin. Ferdinand Enke Verlag, Stuttgart

#### Joseph J

(1931) Nasenplastik und sonstige Geschtplastik nebst Mammoplastik. Curt Kabitzsche, Leipzig

MacComber D W

(1946) Rocky Mountain Medical Journal 43, 466-467

Maliniac J W

(1931) Archives of Otolaryngology 13, 270-274

Matton G, Pickrell K, Huger W & Pound E

(1962) Plastic and Reconstructive Surgery 30, 403-414

Odou B L & Odou E R

(1961) American Journal of Surgery 102, 3-16

Smith A E

(1958) American Journal of Surgery 96, 792-801

Virchow R

(1864) Die Krankhaften Geschwulste. Berlin

Wolfe M M

(1943) Laryngoscope 53, 172-180

Wood J C

(1912) Surgery, Gynecology and Obstetrics 15, 622

# Munchausen's syndrome with multiple pulmonary manifestations<sup>1</sup>

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Fever, haemoptysis, infection and anaemia are recognized in patients with factitious disease (Asher 1951). We report a patient who had all these features in rapid succession during one hospital admission and, in addition, pseudo severe acute asthma requiring mechanical assisted ventilation.

### Case report

A 22-year-old woman, with a 9-year history of asthma was admitted with a severe asthmatic attack. She was distressed and dyspnoeic at rest. Her pulse was 120/minute and her respiration pattern was shallow with few wheezes but coarse crackles at both lung bases. Her peak expiratory flow (PEF) was unrecordable, FEV1 0.11 and FVC 0.31. She had had two previous admissions with acute asthma. During the second of these admissions serial PEF readings were noted to bear little relation to therapy. Major airway obstruction was excluded by bronchoscopy and bronchiectasis by bronchography. She was aggressive and manipulative and had paranoid ideas. As a child she had been beaten by her father and had been taken into care by the Social Services. She was separated from her husband. Her asthma had worsened during her pregnancies - the first

resulting in a stillbirth at 37 weeks, the second in a child handicapped by cerebral palsy, and the third in a premature normal infant.

During the current admission she responded slowly to standard maximal therapy and developed a high (41°C) swinging fever. She insisted on regulating her own nebulizer therapy and drew up each dose with syringe and needle. On the 6th day she became acutely dyspnoeic and distressed, again with an unrecordable PEF, crackles at the left base and a normal chest radiograph. Arterial blood gases on air were: pH 7.44, Pco, 3.5 kPa, Po<sub>2</sub> 13.6 kPa. She continued on maximal therapy for asthma (including aminophylline infusion, intravenous hydrocortisone and antibiotics).

Her distress and dyspnoea worsened and she started to have periods of apnoea of up to 30 seconds duration. She then had a respiratory arrest, was intubated and ventilated. Ventilation was easy and inflation pressures were normal. Four hours later she extubated herself despite heavy sedation. During a subsequent apnoeic attack, her arterial blood gases on air were: pH 7.42, Pco<sub>2</sub> 3.46, Po<sub>2</sub> 7.66. Fifteen minutes after recovery on 35% oxygen they were: pH 7.39, Pco<sub>2</sub> 3.83, Po<sub>2</sub> 23.8. On the 13th day she developed severe left-sided pleuritic chest pain and haemoptysis. The high swinging fever persisted, but when her temperature was measured under close supervision it was always normal. Over the next 10 days the severity of pain, which was refractory to opiates, increased and dramatic haemoptysis continued. These usually occurred before an audience, with profuse coughing, and the bedclothes were frequently spattered with blood. All laboratory investigations were repeatedly normal and her haemoglobin did not fall. At bronchoscopy, fresh

<sup>&</sup>lt;sup>1</sup>Accepted 15 April 1985